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A CLINICAL STUDY OF ONE HUNDRED AND FORTY-TWO CASES OF

HEART-DISEASE

IN CHILDREN.

BY

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[*Reprinted from Archives of Pediatrics, December, 1891.*]

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THIS study is based upon one hundred and thirty-five cases of acquired cardiac disease in children under fourteen years of age, and seven cases of congenital malformation. For about one-third of these I am indebted to Dr. Holt, who has kindly placed at my disposal the record books of the Northwestern Dispensary.

Of these, fifty-one, or thirty-eight per cent., were boys; eighty-four, or sixty per cent., were girls. Of the congenital cases four were boys and three were girls. Their ages are shown by the following table:

Years.....	1	2	3	4	5	6	7	8	9	10	11	12	13	14	
Males.....		1	2	1	4	6	4	1	8	6	5	6	6	1	— 51
Females..		1	3	4	7	9	10	8	9	12	14	3	1	3	— 84
Total...		2	5	5	11	15	14	9	17	18	19	9	7	4	—135

Hence less than nine per cent. occurred under five years, while thirty-eight per cent. occurred under eight years. The ratio of males to females for the whole time was one hundred to one hundred and sixty-four; during the first eight years it was one hundred to one hundred and eighty-eight, and for the next seven years one hundred to one hundred and fifty-one. The preponderance of females, therefore, was greatest before eight years. While the number of males cases was virtually the same each year from nine to fourteen, there was a sudden decrease in the number of female cases between eleven and

twelve. The greatest number occurred at nine, ten, and eleven years, the total for that period being fifty-four, or forty per cent. As the actual date of onset in most of these cases preceded by several months at least the detection of the murmur, these statistics, as well as those of other observers with which they closely agree, would seem to point to the interval between the fifth and twelfth years as the period of greatest danger as regards heart-disease.

The family history regarding rheumatism was obtained in seventy-four cases with the following results:

Father	27 cases,	alone in	17 cases.
Mother	21 "	" "	11 "
Both father and mother			10 "
Brothers or sisters	12,	parents not rheumatic	4 "
Grandparents	25, "	" "	11 "
			53 "
No history of rheumatism			21 "

In seventy-two per cent., therefore, there was a definite family history of rheumatism, or, excluding grandparents, fifty-seven per cent.

In studying the causes of cardiac disease, the possibility of error is very great. Rheumatism is so uncertain in its manifestations in the young that its presence or absence is only to be ascertained by the utmost care. In obtaining a child's history, it seems almost impossible to always arrive at the exact truth. A mild rheumatic attack may have been unnoticed or its occurrence is soon forgotten. I have been impressed with the considerable number of children who have developed rheumatism in whom no rheumatic history could be obtained at the first visit. There is certainly ground for suspicion that in some of these cases there have been previous attacks. The following statistics have been, in all stages, prepared with great care, and I believe are as accurate as such figures can be made.

TABLE

showing history obtained at first visit in one hundred and seventeen cases.

Acute rheumatism	53 cases.
Joint pains or mild subacute rheumatism	25 "
Growing pains without other symptoms	8 "
Total, rheumatic	86 "

Chorea, without growing pains, joint pains, or rheumatism	7 cases.
Measles, without rheumatism or scarlet fever	9 "
Scarlet fever, without rheumatism or measles	5 "
Measles and scarlet fever, without rheumatism	4 "
No previous illness	6 "
Total, non-rheumatic	31 "

Diphtheria is so frequently confused with tonsillitis and the angina of scarlatina that it has not been considered. The total number of cases giving a history of measles was twenty-three, of scarlet fever, twenty-eight. The part played by these two diseases in the production of the cardiac disease was in most instances very uncertain.

Among these thirty-one apparent non-rheumatic patients there appeared, while under observation, rheumatism in eight cases, recurrent tonsillitis in two cases, and chorea without evidence of rheumatism in one case. At the first visit, therefore, eighty-six cases (73.5 per cent.) gave a rheumatic history. Adding to these the cases of rheumatism and tonsillitis developing later, the percentage is raised to 82.1. Erythema, fibrous nodules, and tonsillitis occurred in many cases, but, unless other evidence was also present, they were not classed as rheumatic.

The following intercurrent diseases appeared while these cases were under observation:

Rheumatism	23,	no previous attack in				8
Tonsillitis	9,	"	"	"	"	2
Torticollis	2,	"	"	"	"	2
Purpura	1,	"	"	"	"	1
Keratitis	2,	"	"	"	"	2
Chorea	11,	"	"	"	"	2
Pneumonia	3,	"	"	"	"	3

A history of chorea was obtained in thirty-nine cases, of which thirty-two were distinctly rheumatic. As an intercurrent disease it appeared for the first time in two cases, of which one was rheumatic. It was seen therefore in forty-one cases, or 30.3 per cent., being associated with rheumatism in thirty-three cases. These numbers, it will be observed, are not for all cases of chorea, but for those only with cardiac

lesions. It is a well-known fact that the murmur appearing during chorea sometimes disappears as the choreic movements subside. That occurred in several of these cases. It is in some instances, however, apparent rather than real. In two cases the murmur disappeared as the chorea subsided, but a few weeks later examination showed a distinct mitral regurgitant murmur. In other cases the murmur decreased in intensity, but soon increased and remained as a permanent murmur. Such a condition is probably due to the fresh lighting up of a valvulitis. An attack of rheumatism, perhaps very mild, might readily cause a fresh inflammation on an already sensitive endocardium. While I am strongly inclined to believe that the endocarditis of chorea is in fact rheumatic, in eight cases no history of rheumatism could be obtained, and no evidence of that disease appeared while under observation.

One patient, a girl of three years, gave no history whatever of rheumatism, chorea, measles, scarlet fever, or diphtheria, neither were the parents rheumatic. A murmur developed, or at least increased very markedly, during the course of a severe pneumonia, and remained as a loud, harsh, mitral regurgitant. In another instance, that of a girl nine years of age, there was a family history of rheumatism, but the patient had never shown any evidence of rheumatism and had not had scarlet fever nor diphtheria. During a mild attack of torticollis a blowing mitral murmur developed. This was unquestionably rheumatism.

The clinical history of heart-disease naturally divides itself into three periods,—that of acute inflammation, of compensation, and of heart-failure. For its intelligent management it is necessary that these distinctions should be kept clearly in mind. If the compensation is perfect, as it often is in children to a surprising degree, there will be no symptoms of heart-disease and nothing to treat. All that can be accomplished in any case not suffering from acute inflammation is to establish compensation. If that is already accomplished, it is the height of impropriety to treat the patient for heart-disease. The error must not be made upon the other extreme, however, that the physician has no duty in the case. Such children should be kept under observation both by parents and physician, lest

the condition of compensation be unexpectedly changed to that of heart-failure. Nutrition should be maintained at the highest possible point by diet and properly regulated out-door exercise, and sometimes by the aid of medicine. The child should be especially guarded against exposure to the exanthematous diseases, and above all else should be protected from conditions which may tend to precipitate an attack of rheumatism. Anæmia is a condition full of peril in heart-disease, for, when it is extreme, compensation cannot long be maintained. Yet it is exceedingly common, and is no doubt in many cases a result of the cardiac disease. Record was made upon this point in but seventy-one cases. It was present to a greater or less degree in fifty-nine, in sixteen being noted as extreme.

By far the most common cardiac symptoms were dyspnœa and palpitation. Palpitation was noted in forty-two cases, usually appearing only on exertion. Dyspnœa was but little less common, appearing in thirty-nine cases, in over half being noticed only on exertion. These symptoms were more marked when the aortic valves were at fault. Pain as an urgent symptom appeared in but eleven cases, and was most commonly associated with a mitral obstructive murmur. Cyanosis and œdema, in cases not suffering from acute endocarditis, were extremely rare. Epistaxis was not uncommon, but could hardly be designated as a symptom of heart-disease. The condition so frequently seen in the adult, marked by dyspnœa so extreme as to prevent sleep, tumultuous palpitation, and extreme cardiac distress, cyanosis, and dropsy, is a rare sight in a child under twelve years.

Pitt has drawn attention to a dilatation of the heart which occurs at about the age of fourteen years, even when no valvular lesion is present. It is characterized by anæmia, loss of energy, palpitation, and dyspnœa. The first sound of the heart is faint, and the pulse is weak, rapid, and irregular. This is explained by the great changes which take place in the heart and vessels at this time, especially in girls. From the seventh to the fourteenth year the heart increases very little in actual volume. There is then a period of sudden growth, when the heart increases from eighty to one hundred per cent.,

sometimes within the space of a year or two. This is, therefore, a most critical period and one which requires judicious management. I have in a number of instances seen a cardiac patient progress most satisfactorily until this age was reached, when the whole aspect of the case was changed. Compensation became imperfect, the child grew anæmic and gave the strongest evidence of imperfect nutrition. Development was retarded, though there might be growth in height, the child being thin and without strength or vigor. Sometimes he would develop into a fairly healthy youth, but in other cases, going from bad to worse, would finally succumb. In one case, notably, growth and development seemed to wholly stop at fourteen, and for three years there was a slow and steady retrogression until the girl died. Fortunately, many cases, probably the majority, pass safely through this period, often without perceptible inconvenience. Such patients, as a rule, develop into healthy men and women, and may never show symptoms of cardiac disease.

That endocarditis is sometimes an exceedingly obscure disease was proved by a number of these cases. It sometimes occurs without appreciable symptoms. When accompanying a rheumatic attack there is usually an increase in temperature, or slight fever appears if none has previously been present. The child seems more ill, more so, perhaps, than the arthritis would account for. There may be a peculiar restless, anxious expression with a tendency to cyanosis. The heart's action is disturbed with, perhaps, præcordial distress. The pulse becomes very rapid, this being one of the most constant symptoms. Usually a murmur quickly develops, though some observers have noticed that this sign may be delayed. In the great majority of cases it is mitral regurgitant in character. Sometimes there is a marked reduplication of the second sound heard at the apex, not at the base. This, when present, is very strong evidence of endocardial inflammation. I can confirm the statement that this reduplication is likely to be followed by mitral stenosis. I have seen but one death resulting directly from acute endocarditis. In that case the patient, a girl of nine years, five days after all rheumatic symptoms had disappeared, was not only permitted to leave

her bed against orders, but to go into the street, where she died suddenly.

In preparing this paper all doubtful cases have been rejected. One well-known author has included in his statistics "cases of thick sounds, thumping action, displaced heaving impulse." Such cases have been rejected, as the pathological conditions present are, to say the least, very problematical. The following murmurs were heard:

Mitral regurgitation	in	124	cases,	alone	in	93	cases.
Mitral obstruction	"	16	"	"	"	4	"
Aortic regurgitation	"	9	"	"	"	0	"
Aortic obstruction	"	26	"	"	"	3	"
Double mitral and double aortic					"	2	"
Mitral regurgitation and double aortic					"	3	"
Mitral regurgitation and aortic obstruction					"	18	"
Double aortic (alone)					"	1	"

In four cases murmurs were heard, but were not differentiated. At the first examination four patients were suffering from acute primary endocarditis. Of these one died, two developed a mitral regurgitant murmur, and one a double mitral murmur. The statement that a soft blowing murmur is recent, while a loud harsh or musical murmur is old, is usually quite true; but it is a rule subject to many exceptions.

The following peculiarities in physical signs were noted:

1. The apex lies higher and more to the left than in the adult, being outside the nipple-line until four years and rarely inside until eight years.

2. The area of dulness is comparatively large, so that the normal heart may, without caution, be considered as hypertrophied.

3. The cardiac impulse is more clearly visible and can be felt more distinctly than in the adult.

4. Murmurs are heard over a comparatively wide area, especially the mitral regurgitant, which is not infrequently heard over the whole chest.

5. The rate may be readily increased and the rhythm disturbed by slight causes, so that in the examination of a nervous child rapidity and irregularity must be considered of very little importance.

One hundred and twenty-four patients presented clinical evidence of mitral regurgitation. If, as Sansom alleges, in pericarditis a murmur may be heard indistinguishable from the systolic murmur of mitral regurgitation, these were, perhaps, not all due to insufficiency of the mitral valves. The great frequency of the mitral murmur is beyond doubt, by this term being meant a systolic murmur heard with greatest intensity at the apex and conveyed to the left, either into the axilla or to the back. Such a murmur occurred in 91.7 per cent. of these cases.

The murmur of most interest or certainty, the one that has been the subject of most discussion, is the presystolic mitral. In one hundred and one cases it was noted sixteen times, being in relation to the mitral regurgitant as 1 to 7.75. Rheumatism as a factor in its production was less strongly marked than in either of the other murmurs. In four cases no history whatever of rheumatism or chorea was obtained in patient or family. Definite rheumatic history was obtained in seven cases, consisting simply of growing pains in two. In one case chorea developed later and in one acute rheumatism. Fifty-six per cent., therefore, gave a personal rheumatic history, the percentage for all cases being eighty-two. In the remaining cases the presence of rheumatism was somewhat uncertain. These facts bear out the statement of Sansom, that mitral stenosis is intimately associated with rheumatism, but most frequently with insidious varieties. The same fact has been observed by Chapin.

Symptoms, as in all varieties of valvular disease, were often obscure and at times entirely absent. They were, however, more marked than in simple mitral regurgitation. Pain was more common than with any other lesion, and dyspnœa on exertion was the rule. Palpitation was also common, while bronchitis and cough was more constant than in any other form of valvular disease.

It is universally agreed that the presystolic murmur is rare in infancy. The youngest of these patients was five years old; eleven were over eight years, six being between eight and ten. As far as I have observed, it is slow in its development, never appearing suddenly as does the mitral regurgitant. The

character of the abnormal sound is subject to change from time to time, more so than any other murmur. It may become very faint or even imperceptible, but I have not seen it disappear permanently. It is very sure to return, and hopes based on its disappearance are almost certain to be disappointed. There is frequently no perceptible cause for this changeability. Tonic treatment seems to have but little effect in causing permanent disappearance of the murmur.

The mitral obstructive is probably more frequently overlooked than any other murmur, yet it is quite distinct and characteristic. It is, as a rule, harsh and of a rattling, blubbering or wheezing character. It differs decidedly from other murmurs in one particular. Instead of rising to a maximum and then gradually decreasing or shading off into silence, it rises rapidly to the maximum and suddenly ceases as the apex strikes the chest-wall. Its area of diffusion is, also, very limited. As the stethoscope is carried from the apex, a point is quickly reached at which the murmur suddenly and completely ceases. If a regurgitant murmur is also present and the heart is acting rapidly, the two murmurs may run so closely together as to be with difficulty separated. In that case the first portion, or obstructive murmur, suddenly ceases at a given point, while the regurgitant remains unchanged. If the second sound is reduplicated at the apex, the certainty of mitral stenosis is increased. A thrill is by no means as common as in the adult. It is sometimes absent in well-marked cases. It was noted in but seven cases, but may have occurred in other instances.

One case, that of a girl of eight years, is of interest, in showing the order of development of the murmurs. When first seen there was a mitral regurgitant murmur, dating, no doubt, from an attack of diphtheria, which had been followed by renal symptoms. Two years later subacute rheumatism developed, and during the next two years was followed by several well-marked attacks with nodules. Four years after the first examination a mitral obstructive murmur was heard, and a few months later a thrill appeared, followed the next year by an aortic obstructive murmur, and this in turn by an aortic regurgitation.

Another patient, a boy six years old, was brought to the Polyclinic in April last. There was no history whatever of rheumatism or chorea either in patient or family. There were slight joint pains and pneumonic consolidation was detected at the left base. The heart-action was tumultuous, but no abnormal sound could be positively detected. Four days later there was unquestioned endocarditis and distinct pericardial friction sounds. The acute symptoms disappeared during the next two weeks, a mitral regurgitant murmur being left. He was not seen again until August, when a mitral regurgitant murmur was heard, and also a loud blubbering mitral obstruction, accompanied by a slight thrill. The boy was in excellent physical condition, and was supposed by the parents to be in perfect health.

The aortic murmurs were much more definitely associated with rheumatic histories than were the mitral. In the twenty-six aortic cases the family history was taken in seventeen, in twelve of which it was definitely rheumatic. In fourteen there was a clear history of articular rheumatism, and in four of indefinite and growing pains. In but three could a positive negative history be obtained. Nine cases were choreic. Nearly seven cases in eight gave a personal history of chorea or rheumatism. Two of the non-rheumatic cases had had scarlet fever about one year before, from which it is quite possible the cardiac lesions resulted.

While symptoms were in many cases absent or very obscure, they were as a rule somewhat more distinctive than in mitral regurgitation. Anæmia was almost constantly present; dyspnœa was noted fifteen times and palpitation fourteen. Both symptoms were more continuous, depending less upon exertion than with the mitral lesions. Pain was prominent in but three cases not associated with mitral stenosis. Cases of double aortic murmur were invariably marked by palpitation and dyspnœa, at least on exertion. Dyspnœa was by far the most constant and definite symptom.

In but few instances did the murmur wholly disappear, though a change in character was not uncommon, a loud, harsh murmur becoming soft and blowing, and at times difficult of detection. The two murmurs that disappeared were

slow in doing so, two years being required in one case and three and a half in the other. In one patient, a girl of nine years, who gave a history of chorea and rheumatism, there was a mitral regurgitant and loud obstructive murmur. During a period of four years she had several attacks of subacute rheumatism, but at the same time the aortic obstructive murmur gradually disappeared, while an aortic regurgitant developed. For over two years there has been no rheumatism. The murmurs remain, but are unaccompanied by symptoms. The girl has grown large and strong, and is apparently well.

Study of these cases tends to show that if intercurrent attacks of rheumatism can be prevented, the progress of aortic disease is not especially worse than that of mitral. That aortic disease is, however, a matter of great seriousness cannot be doubted. It is the result of an extensive endocarditis, and is in a large majority of cases closely associated with rheumatism. Children suffering from this form of valvular disease are especially subject to recurrent attacks of rheumatism with accompanying endocarditis. Taking this into consideration, the prognosis of aortic disease is far worse than that of mitral disease. While it is possible that the murmur may disappear as the child develops, the probability of such a fortunate outcome is very small.

Seven cases gave evidence of congenital disease or malformation. Space forbids extended discussion of this subject, and the pathological conditions are so varied that classification of symptoms or physical signs is impossible. I simply subjoin, therefore, a brief history of each case. A point to me of especial interest has been with reference to bronchitis and its possible action, by obstructing pulmonary circulation, in causing continued patency of the fœtal openings.

Case I.—A. P., four months old. Gave no history of cyanosis or other cardiac symptoms. Has always been weak and puny and subject to bronchitis. Was suffering from pneumonia, from which he died the following day. At the autopsy nothing abnormal was found at either valvular orifice. The foramen ovale was open, being the size of a lead-pencil, with smooth, rounded margins. It was, however, valvular in char-

acter, and probably allowed very little regurgitation. The ductus arteriosus was also open, the aorta being slightly contracted between it and the heart.

Case II.—J. A., two years old; feeble and poorly nourished; has never walked; had always been subject to bronchitis, and had been cyanotic from birth. A very loud systolic murmur was heard, being most intense at the left of the sternum at the juncture of the fourth rib, and transmitted towards the left shoulder. This case was seen by competent diagnosticians, who agreed in the diagnosis of pulmonary stenosis with open foramen ovale.

Case III.—S. O., two years old. When first brought for treatment was suffering from broncho-pneumonia, from which she recovered. History of cyanosis doubtful. A harsh systolic murmur was heard over the whole left side of the chest anteriorly. The point of the maximum intensity was well above the apex, to the left of the sternum, the bruit being transmitted towards the left shoulder. The certainty as to its congenital nature was, perhaps, less in this than in any of the other cases.

Case IV.—T. K., three months old. Cyanosis marked while crying or excited, little or none when quiet. Systolic murmur heard loudest at second or third space at left of sternum, audible over whole chest, but more distinct towards the left shoulder than at the apex. The child died at the age of four months. The heart was found at the autopsy to be normal in size. Immediately beneath the aortic valve and above the muscular substance of the ventricular septum was an opening one-fifth of an inch in diameter, which communicated with the right ventricle, the aortic valves and coronary arteries being normal. This opening entered the ventricle at a considerable distance from the pulmonary orifice, at which there was marked stenosis, the right ventricle being greatly hypertrophied. The pulmonary artery was of normal size, and the valve was somewhat thickened and adherent, probably sufficient to allow regurgitation during life. The foramen ovale and ductus arteriosus were closed. The inter-ventricular opening was chiefly peculiar from its position, which was unusually high.

Case V.—J. B., eleven months old. No history of illness except bronchitis, to which she was very subject. Cyanosis had not been observed. There was a blowing, systolic murmur, most distinct at the third space left of the sternum, conveyed upward and outward, but audible over a large area. During two and a half years the child has grown naturally, and has had no illness, except measles and an occasional bronchitis, which is never severe, but very rebellious to treatment. The murmur remains unchanged.

Case VI.—C. B., three years old. No history of rheumatism or chorea in patient or family. Patient had never been strong, but had had no illness except bronchitis. No cyanosis was noticed till child was several months old, when it gradually appeared, and had markedly increased during the past year. When quiet it was scarcely perceptible, but upon exertion it became extreme. A loud, harsh murmur was heard just before and with the first sound, the point of maximum intensity being above the apex near the sternum. It was conveyed upward and to the left, but was heard distinctly at the apex. After two years the cyanosis is not quite as marked, but the murmur is unchanged. The boy is growing, but shows marked clubbing of the finger-ends.

Case VII.—A. B., ten months old. Child was puny; has had bronchitis much of the time, but not severely. Cyanosis upon crying was marked, and had been noticed since birth. A loud, blowing murmur, systolic and probably diastolic, was heard over the whole chest; of maximum intensity at the left of the sternum above the apex.